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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/928,872	08/13/2001	Richard Kolesnick	6923-106	8412

20583 7590 09/10/2002

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EXAMINER	
HUYNH, PHUONG N	

ART UNIT	PAPER NUMBER
1644	8

DATE MAILED: 09/10/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No. 09/928,872	Applicant(s) KOLESNICK ET AL.	
	Examiner " Neon" Phuong Huynh	Art Unit 1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE ____ MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *12 June 2002* .

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-3,5,7 and 9-13* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-3,5,7 and 9-13* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 7 .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

1. Claims 1-3, 5, 7 and 9-13 are pending.

2. In view of the amendment filed 6/12/02, the following rejections remain.

3. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. Claims 1-3, 5, 7, 9-13 stand rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention.

The specification does not provide adequate written description of the claimed invention. The legal standard for sufficiency of a patent's (or a specification's) written description is whether that description "reasonably conveys to the artisan that the inventor had possession at the time of the ...claimed subject matter", Vas-Cath, Inc. V. Mahurkar, 19 USPQ2d 1111 (Fed. Cir. 1991). In the instant case, the specification does not convey to the artisan that the Applicant had possession at the time of invention of the claimed methods employing *any* **chemotherapeutic stress stimulus** to induce apoptosis and thereby identifying compounds that increase or decrease a cell's sensitivity to acid sphingomyelinase activity such as apoptosis, sphingomyelin and ceramide levels.

The specification discloses methods for identifying a compound mentioned above employing *only* **radiation stress stimulus** to induce apoptosis and thereby identifying compounds that increase or decrease a cell's sensitivity to acid sphingomyelinase activity such as apoptosis morphology, sphingomyelin and ceramide levels. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, Applicant was not in possession of the claimed genus. *See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.*

Applicant is directed to the Revised Interim Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

Applicants' arguments filed 6/12/02 have been fully considered but are not found persuasive.

Applicants' position is that the support the claimed phrase "chemotherapeutic stress stimulus" can be found throughout the specification on page 1, line 10, page 3, line 19; page 4, line 13; page 14, line 22 and page 38, line 7 where "chemotherapeutic agent-induced apoptosis", "chemotherapeutic therapies" and "chemotherapeutic agents" are mentioned.

However, the phrase "chemotherapeutic stress stimulus" has no support in the specification. There is no structure associated with the term "chemotherapeutic stress stimulus". Further, the specification discloses only a method for identifying a compound employing *only* **radiation stress stimulus** to induce apoptosis and thereby identifying compounds that increase or decrease a cell's sensitivity to acid sphingomyelinase activity such as apoptosis morphology, sphingomyelin and ceramide levels. The specification merely mentioned "chemotherapeutic agents". Given the lack of any additional species of "chemotherapeutic stress stimulus", one of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, Applicant was not in possession of the claimed genus. *See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.*

5. Claims 1-3, 5, 7, 9-13 stand rejected under 35 U.S.C. 112, first paragraph, containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. **This is a new matter rejection**.

The "chemotherapeutic stress stimulus" in Claims 1-3, 5, 7, 9-13 represents a departure from the specification and the claims as originally filed. The passages pointed out by applicant in the amendment filed 12/14/01 do not provide a clear support for the said phrase.

Applicants' arguments filed 6/12/02 have been fully considered but are not found persuasive.

Applicants' position is that the support the claimed phrase "chemotherapeutic stress stimulus" can be found throughout the specification on page 1, line 10, page 3, line 19; page 4,

line 13; page 14, line 22 and page 38, line 7 where "chemotherapeutic agent-induced apoptosis", "chemotherapeutic therapies" and "chemotherapeutic agents" are mentioned.

However, the phrase "chemotherapeutic stress stimulus" has no support in the specification.

6. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 103(a) that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

7. This application currently names joint inventors. In considering Patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

8. Claims 1-3, 5, 7 and 9-13 stand rejected under 35 U.S.C. 103(a) as being unpatentable over Lowe *et al* (Cell 74: 957-967, Sept 1993; PTO 1449) in view of Jarvis *et al* (Proc. Natl. Acad Sci USA: 91: 73-77, Jan 1994: PTO 1449), Cifone *et al* (EMBO J 14(23): 5859-68, 1995; PTO 1449) and US Pat No 5,773,278 (June 1998, PTO 892) or Horinouchi *et al* (Nature Genetics 10: 288-293, July 1995; PTO 1449) or Otterbach *et al* (Cell 81: 1053-61, June 1996; PTO 1449).

Lowe *et al* teaches a method for identifying compound which increases or decreases a cell's sensitivity to p53-mediated apoptosis comprising contacting p53 deficient cells (p53-/-) and p53 positive cells (p53+/- and p53+/+) with a test compound such as chemotherapeutic agents 5-Fluorouacil, etoposide, adriamycin, and sodium azide (See Table 1, page 958, Fig 5, in particular) to induce apoptosis (See Figs 2-6, page 965, Experimental procedure, in particular) wherein the apoptotic morphology comprises cellular condensation, nuclear condensation or zeiosis (See page 960, column 2, first full paragraph, Fig 6, in particular).

The claimed invention in claim 1 differs from the reference only by the recitation of contacting an acid sphingomyelinase-deficient cell and if the cell exposed to chemotherapeutic agent exhibits more severe apoptotic morphology than the control, the test compound represents a compound, which increases a cell's sensitivity to acid sphingomyelinase-related apoptosis.

The claimed invention in claim 2 differs from the reference only by the recitation of contacting an acid sphingomyelinase-deficient cell and if the sphingomyelin is decrease while the level of ceramide increases in cell exposed to chemotherapeutic agent as compared to the control, the test compound represents a compound, which increases a cell's sensitivity to acid sphingomyelinase-related apoptosis.

The claimed invention in claims 3 differs from the reference only by the recitation of the acid sphingomyelinase-deficient cell is part of a genetically engineered nonhuman animal deficient for acid sphingomyelinase gene.

The claimed invention in claims 5 differs from the reference only by the recitation of the cell exhibiting acid sphingomyelinase activity with a test compound, exposing said cells to a chemotherapeutic stress stimulus, comparing the levels of sphingomyelin and ceramide and if the sphingomyelin level is greater while ceramide level is less than the control, the test compound represents a compound which decreases a cell's sensitivity to acid sphingomyelinase-related apoptosis.

The claimed invention in claims 7 differs from the reference only by the recitation of the cell is part of a genetically engineered nonhuman animal deficient in endogenous acid sphingomyelinase gene activity and containing a functional human acid sphingomyelinase transgene capable of expressing functional human acid sphingomyelinase.

The claimed invention in claims 9 differs from the reference only by the recitation of the apoptotic morphology comprises cellular condensation, nuclear condensation and zeiosis.

The claimed invention in claims 10 and 11 differs from the reference only by the recitation of the acid sphingomyelinase-deficient cells are part of cell lines or a genetically engineered nonhuman animal deficient for the acid sphingomyelinase gene.

The claimed invention in claims 12 differs from the reference only by the recitation of the transgenic cells that are deficient in endogenous acid sphingomyelinase gene activity and contain a functional human acid sphingomyelinase gene.

The claimed invention in claims 13 differs from the reference only by the recitation of the cells are genetically engineered cells that exhibit greater level of acid sphingomyelinase activity than non-genetically engineered cells of the same type.

Jarvis *et al* teach when cells such as HL60 and U937 that exhibiting acid sphingomyelinase activity are exposed to various chemotherapeutics stress such as sphingomyelinase and C8ceramide, the cells undergo apoptosis (See entire document, Figs 1, 3 and 6, in particular). Jarvis *et al* teach how to determine the morphological features of apoptosis such as cellular condensation, nuclear condensation or zeiosis (See Fig 6, Materials and Methods, in particular).

Cifone *et al* teach when cells such as HuT78 that exhibits acid sphingomyelinase activity are exposed to chemotherapeutics stress stimulus such as crosslinking Fas receptor using anti-Fas antibody or TNF, apoptotic cell death results. This is associated with a decrease in the level of sphingomyelin (breakdown) with a concomitant increase in the level of ceramide (generation) (See Figs 2-4, 7, page 5865-5866, Materials and methods, in particular). Cifone *et al* teach how to measure the levels of ceramide and sphingomyelin (See page 5866, column 1, in particular). Cifone *et al* teach that it is of interest to screen for compound which increase or decrease the cell's sensitivity to acid sphingomyelinase related apoptosis such as measuring the levels of ceramide and sphingomyelinase activity (See page 5865, column 2, Biological implications, in particular).

The '278 patent teaches acid sphingomyelinase deficient cell and cell line such as fibroblast or lymphoblasts generated from Niemann-Pick disease (NPD) patient and transgenic mice overexpressing the human acid sphingomyelinase gene (See column 27, lines 61-67, column 34, lines 17-30, in particular). The '278 patent teaches nucleotide encoding for human acid sphingomyelinase (ASM) is useful for engineering transgenic mice and cell lines overexpressing the human ASM for screening compound for treatment of Niemann-Pick disease (See column 7, lines 31-43, column 24, lines 46-58, in particular).

Horinouchi *et al* teach acid sphingomyelinase deficient mice as a model for type A and B human Niemann-Pick disease (See entire document, Methods, in particular).

Otterbach *et al* teach acid sphingomyelinase deficient mice as a model for the neurovisceral form of human Niemann-Pick disease (See entire document, Experimental Procedure, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the p53 deficient cells for a method for identifying compound which increase or decrease a cell's sensitivity to apoptosis to p53 as taught by Lowe *et al* for the acid sphingomyelinase deficient cells wherein the cells are part of the cell lines or genetically engineered transgenic mouse or cell lines expressing or overexpressing the human ASM as taught by the '278 patent or the genetically engineered mice deficient for the acid sphingomyelinase as taught by Horinouchi *et al* or Otterbach *et al* for a method for identifying compound which increases or decreases a cell's sensitivity to sphingomyelinase-related apoptosis as taught by Cifone *et al* and Jarvis et al. From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable of success in producing the claimed invention.

One having ordinary skill in the art at the time the invention was made would have been motivated to do this because Jarvis *et al* teach acid sphingomyelinase induces cell death by apoptosis in cells exhibiting acid sphingomyelinase activity (See entire document, Figs 1, 3 and 6, in particular). Cifone *et al* teach that it is of interest to screen for compound which increase or decrease the cell's sensitivity to acid sphingomyelinase related apoptosis (See page 5865, column 2, Biological implications, in particular). The '278 patent teaches that Niemann-Pick disease (NPD) is associated with acid sphingomyelinase deficiency and human acid sphingomyelinase (ASM) transgenic mice and cell lines overexpressing the human ASM is useful for screening compound for treatment of Niemann-Pick disease (See column 7, lines 31-43, column 24, lines 46-58, in particular). Horinouchi *et al* teach acid sphingomyelinase deficient mice as a model for type A and B human Niemann-Pick disease (See entire document, Methods, in particular). Otterbach *et al* teach acid sphingomyelinase deficient mice is a useful model for the neurovisceral form of human Niemann-Pick disease (See entire document, Experimental Procedure, in particular).

Applicants' arguments filed 6/12/02 have been fully considered but are not found persuasive.

Applicants' position is that (1) Lowe does not teach methods to identify any compounds; (2) the chemotherapeutic compounds such as 5-fluoroaucil, etoposide, and adriamycin are chemotherapeutic stress stimuli and not test compounds as taught by Lowe; (3) the chemotherapeutic agents mentioned above did not increase or decrease a cell's sensitivity to p53-mediated apoptosis; (4) Javis et al teach neutral sphinogmyelinase and not acid sphingomyelinase

in the sphingomyelinase pathway; (5) Clifone et al does not teach the term "linking Fas receptor using anti-Fas antibody or TNF constitutes "chemotherapeutic stress stimulus"; (6) Cifone speculates that although "tumor- or virus-transformed cells are likely to develop strategies to block Fas/APO-1-generated apoptotic signals, unraveling key steps of the Fas/APO-1 apoptotic pathway, should indicate possible targets for these strategies; (7) Schuman et al does not teach or suggest a method for identifying compounds that modulate acid sphingomyelinase-related apoptosis; (8) Horinouchi et al does not teach or suggest a method for identifying compounds that modulate acid sphingomyelinase-related apoptosis by exposing certain cells to a chemotherapeutic stress stimulus, as recited in all pending claims and (9) Otterbach et al does not teach or suggest a method for identifying compounds that modulate acid sphingomyelinase-related apoptosis by exposing certain cells to a chemotherapeutic stress stimulus, as recited in all pending claims.

In response to applicant's arguments against the references individually, one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986). Further, the specification does not define the term "chemotherapeutic stress stimulus"; the "test compound" is any chemotherapeutic agent, which is the same as "chemotherapeutic stress stimulus" as defined on page 1, line 10, page 3, line 19; page 4, line 13; page 14, line 22 and page 38, line 7 where "chemotherapeutic agent-induced apoptosis", "chemotherapeutic therapies" and "chemotherapeutic agents" are mentioned.

Lowe *et al* teaches a method for identifying compound which increases or decreases a cell's sensitivity to p53-mediated apoptosis comprising contacting p53 deficient cells (p53-/-) and p53 positive cells (p53+/- and p53+/+) with a test compound such as chemotherapeutic agents 5-Fluorouacil, etoposide, adriamycin, and sodium azide (See Table 1, page 958, Fig 5, in particular) to induce apoptosis (See Figs 2-6, page 965, Experimental procedure, in particular) wherein the apoptotic morphology comprises cellular condensation, nuclear condensation or zeiosis (See page 960, column 2, first full paragraph, Fig 6, in particular).

The claimed invention differs from the reference only by the recitation that the method for identifying a compound using acid sphingomyelinase-deficient cell, genetically engineered nonhuman animal deficient in endogenous acid sphingomyelinase, transgenic cells deficient in endogenous acid sphingomyelinase activity, or genetically engineered cells that exhibit a greater

level of sphingomyelinase activity, monitoring the exposed cells to chemotherapeutic stress stimuli in the presence or absence of test compound for the presence of an apoptotic morphology, and comparing the levels of sphingomyelin and ceramide present in the exposed cells.

Horinouchi *et al* teach acid sphingomyelinase deficient mice as a model for type A and B human Niemann-Pick disease (See entire document, Methods, in particular).

Otterbach *et al* teach acid sphingomyelinase deficient mice as a model for the neurovisceral form of human Niemann-Pick disease (See entire document, Experimental Procedure, in particular).

The '278 patent teaches acid sphingomyelinase deficient cell and cell line such as fibroblast or lymphoblasts generated from Niemann-Pick disease (NPD) patient and transgenic mice overexpressing the human acid sphingomyelinase gene (See column 27, lines 61-67, column 34, lines 17-30, in particular). The '278 patent teaches nucleotide encoding for human acid sphingomyelinase (ASM) is useful for engineering transgenic mice and cell lines overexpressing the human ASM for screening compound for treatment of Niemann-Pick disease (See column 7, lines 31-43, column 24, lines 46-58, in particular).

Jarvis *et al* teach how to determine the morphological features of apoptosis such as cellular condensation, nuclear condensation or zeiosis (See Fig 6, Materials and Methods, in particular). Jarvis *et al* further teach when cells such as HL60 and U937 that exhibiting acid sphingomyelinase activity are exposed to various chemotherapeutics stress such as sphingomyelinase and C8ceramide, the cells undergo apoptosis (See entire document, Figs 1, 3 and 6, in particular).

Cifone *et al* teach when cells such as HuT78 that exhibits acid sphingomyelinase activity are exposed to chemotherapeutics stress stimulus such as crosslinking Fas receptor using anti-Fas antibody or TNF, apoptotic cell death results. This is associated with a decrease in the level of sphingomyelin (breakdown) with a concomitant increase in the level of ceramide (generation) (See Figs 2-4, 7, page 5865-5866, Materials and methods, in particular). Cifone *et al* teach how to measure the levels of ceramide and sphingomyelin (See page 5866, column 1, in particular). Cifone *et al* teach that it is of interest to screen for compound which increase or decrease the cell's sensitivity to acid sphingomyelinase related apoptosis such as measuring the levels of ceramide and sphingomyelinase activity (See page 5865, column 2, Biological implications, in particular).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to substitute the p53 deficient cells as taught by Lowe *et al* for the acid sphingomyelinase deficient cells wherein the cells are part of the cell lines or genetically engineered transgenic mouse or cell lines expressing or overexpressing the human ASM as taught by the '278 patent or the genetically engineered mice deficient for the acid sphingomyelinase as taught by Horinouchi *et al* or Otterbach *et al* for a method for identifying compound which increases or decreases a cell's sensitivity to sphingomyelinase-related apoptosis by monitoring the exposed cells for the presence of apoptotic morphology and sphingomyelinase activity as taught by Jarvis et al and monitoring the levels of sphingomyelin and ceramide as taught by Cifone and Jarvis et al. From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable of success in producing the claimed invention.

One having ordinary skill in the art at the time the invention was made would have been motivated to do this because Jarvis *et al* teach acid sphingomyelinase induces cell death by apoptosis in cells exhibiting acid sphingomyelinase activity (See entire document, Figs 1, 3 and 6, in particular). Cifone *et al* teach that it is of interest to screen for compound which increase or decrease the cell's sensitivity to acid sphingomyelinase related apoptosis (See page 5865, column 2, Biological implications, in particular). The '278 patent teaches that Niemann-Pick disease (NPD) is associated with acid sphingomyelinase deficiency and human acid sphingomyelinase (ASM) transgenic mice and cell lines overexpressing the human ASM is useful for screening compound for treatment of Niemann-Pick disease (See column 7, lines 31-43, column 24, lines 46-58, in particular). Horinouchi *et al* teach acid sphingomyelinase deficient mice as a model for type A and B human Niemann-Pick disease (See entire document, Methods, in particular). Otterbach *et al* teach acid sphingomyelinase deficient mice is a useful model for the neurovisceral form of human Niemann-Pick disease (See entire document, Experimental Procedure, in particular).

In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the

applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

9. No claim is allowed.

10. **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a).

Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a). A shortened statutory period for response to this final action is set to expire THREE MONTHS from the date of this action. In the event a first response is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event will the statutory period for response expire later than SIX MONTHS from the date of this final action.

11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to "Neon" Phuong Huynh whose telephone number is (703) 308-4844. The examiner can normally be reached Monday through Friday from 9:00 am to 6:00 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist whose telephone number is (703) 308-0196.

12. Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform to the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 305-7401.

Phuong N. Huynh, Ph.D.
Patent Examiner
Sept 9, 2002

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